Acute left ventricular failure after bilateral lung transplantation for idiopathic pulmonary arterial hypertension

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Video clip is available online.

We report a case of unexpected severe left ventricular (LV) dysfunction in a patient undergoing a bilateral lung transplant (BLTx). We attribute his complicated postoperative course to diastolic dysfunction in a chronically deprived LV in combination with an increased preload, caused by normalization of the pulmonary vascular resistance (PVR), and an increased afterload during weaning.

CLINICAL SUMMARY

After optimal medical treatment for 12 years, a 23-year-old man with idiopathic pulmonary arterial hypertension had deterioration to New York Heart Association functional class III. Catheterization indicated elevated pulmonary arterial pressure (PAP) values of 121 mm Hg systolic, 72 mm Hg diastolic, and 89 mm Hg mean; a severely reduced cardiac index of 1.63 L/(min · m^2); and a PVR of 2309 dynes · s · cm^{-5}. In January 2012, the patient underwent an urgent BLTx because of uncontrollable right ventricular failure (Figure 1, *A*). Surgery was uneventful. After the operation, PAP decreased to 30 mm systolic, 20 mm diastolic, and 22 mm Hg mean. PVR decreased to 204 dynes · s · cm^{-5}. Oxygenation, hemodynamics, and chest radiography were normal. Three hours after extubation, at the end of postoperative day 1, we observed high systemic pressure (150 mm Hg systolic, 87 mm Hg diastolic, and 108 mm Hg mean), cardiac index (5.3 L/[min · m^2]), and PAP (82 mm Hg systolic, 43 mm Hg diastolic, and 55 mm Hg mean). One hour later, the patient collapsed (Figure 2) and was immediately resuscitated. Return of spontaneous circulation was achieved within 4 minutes. Echocardiography revealed LV akinesia (Figure 1, *B*). Because of persistent hemodynamic and respiratory instability, venoarterial extracorporeal membrane oxygenation was initiated. Within 36 hours, lactate level had normalized, pulmonary edema had resolved, and epinephrine infusion was discontinued. On postoperative day 6, transthoracic echocardiography confirmed LV recovery, and the extracorporeal membrane oxygenation was discontinued the same day. LV function improved further and was normalized on postoperative day 20, at which time the patient was discharged from the intensive care unit. Five months after BLTx, cardiac and pulmonary functions were acceptable.

DISCUSSION

On the basis of the LV ejection fraction on repeated transthoracic echocardiography before BLTx, we were unable to predict the complicated postoperative course of this patient. In cases of pretransplant LV dysfunction, there is often a relation to right ventricular dysfunction in patients with pulmonary hypertension, possibly through ventricular interdependence.[1] Our patient, however, had a normal LV ejection fraction (60%). Reperfusion edema, pulmonary rejection, and infection were considered unlikely causes for the acute deterioration on the basis of the time course of the deterioration (Figure 2), the absence of sputum, the clear chest radiography, the stable saturation values before collapse, and the severe LV dysfunction.

We believe that the mechanism leading to LV failure after extubation was the normalization of preload in a chronically preload-deprived LV. Normalization of the PVR through replacement of the entire pulmonary vascular bed must have led to an increased LV preload, as well as a rightward shift of the dyskinetic interventricular septum (Figure 1, *B*). The LV of this young man, who had a history of PH since childhood, never had been exposed to such a preload before. LV diastolic dysfunction emerged and came to full expression with an increased afterload in the awake patient immediately after extubation.

A stroke volume of 34 mL, calculated from pretransplant right heart catheterization, and a small LV cavity on transthoracic echocardiography confirm our hypothesis.

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Disclosures: Authors have nothing to disclose with regard to commercial support.
Received for publication July 6, 2012; revisions received Sept 12, 2012; accepted for publication Oct 11, 2012; available ahead of print Nov 12, 2012.

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J Thorac Cardiovasc Surg 2013;145:e7-9
0022-5223/$36.00

http://dx.doi.org/10.1016/j.jtcvs.2012.10.020

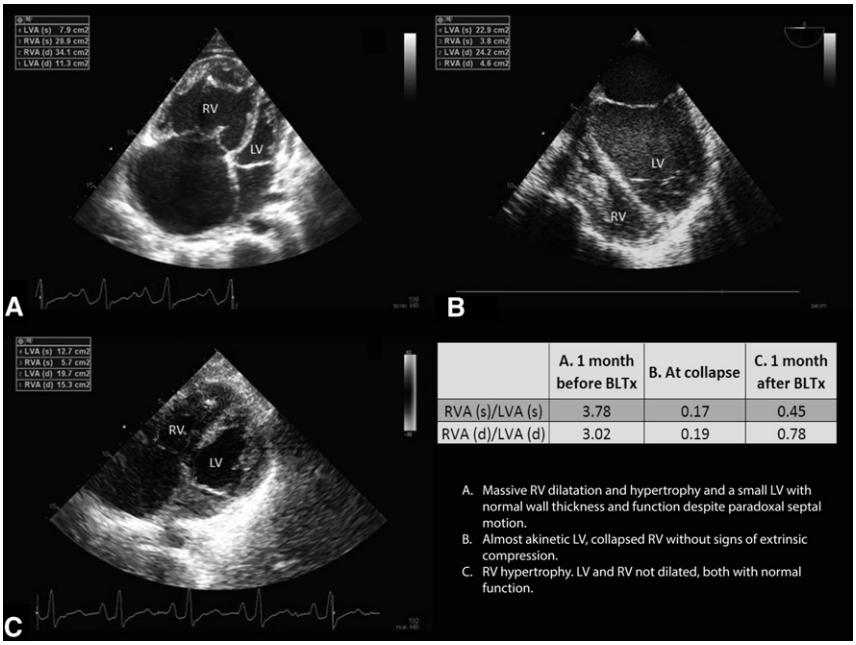

FIGURE 1. Echocardiographic images 1 month before bilateral lung transplant (*BLTx*; A), at collapse (B), and 1 month after lung transplant (C). *LV,* Left ventricle; *LVA (d)*, diastolic left ventricular area; *LVA (s)*, systolic left ventricular area; *RV,* right ventricle; *RVA (d)*, diastolic right ventricular area; *RVA (s)*, systolic right ventricular area.

In retrospect, there were 2 interventions that could perhaps have prevented this course of events. First, an atrial septal defect could have been created before transplant, enabling right-to-left shunting. Currently, atrial septal defect creation is only considered in patients with pulmonary hypertension who have persistent right ventricular failure despite maximal therapy, and the procedure is often performed as a bridge to transplant.[2] The resulting increase in LV preload might have offered a training period for the LV before BLTx. Still, the correct atrial septal defect size and the timing of the procedure remain highly debatable.[3]

Second, the weaning process should have been more controlled, to allow adaptation to the altered hemodynamic

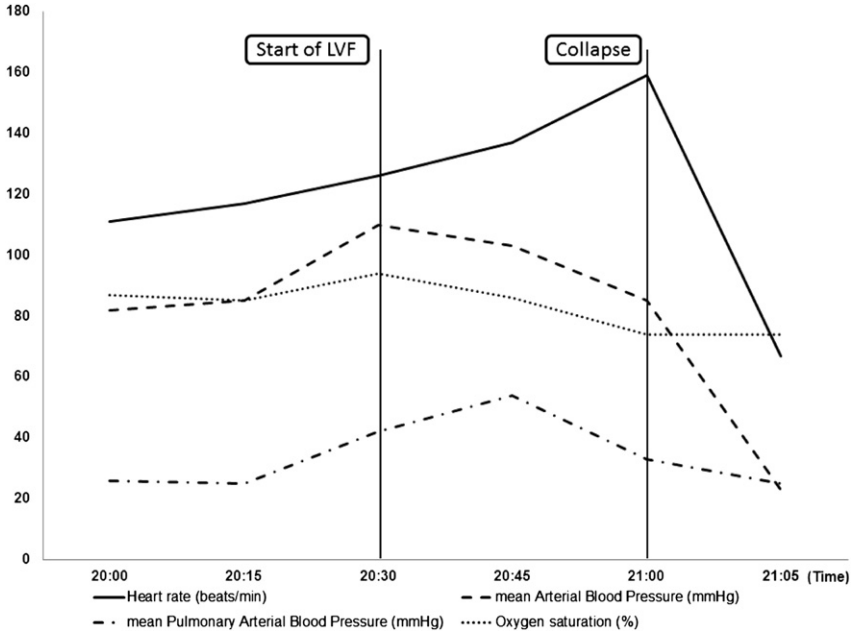

FIGURE 2. Hemodynamics during the hour before collapse. *LVF,* Left ventricular failure.

status.[4] Determination of the exact timing, however, remains difficult. Monitoring of PAP, pulmonary capillary wedge pressure, and LV function before and during the weaning process is mandatory.[4]

We report the case of a young man with idiopathic pulmonary arterial hypertension who underwent urgent BLTx for decompensation. We attribute his complicated postoperative course to diastolic dysfunction in a chronically deprived LV in combination with an increased preload, resulting from normalization of the PVR, and an increased afterload during weaning. Awareness of this problem may lead to earlier discrimination of such patients and a more controlled weaning process after BLTx.

We thank Piet Claus and Maja Cikes for their valuable help regarding the analysis of echocardiographic images.

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Esophageal complications of catheter ablation for atrial fibrillation: A case report

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Atrial fibrillation is the most common cardiac arrhythmia. It is commonly known as irregularly irregular because the time elapsing between 2 consecutive R waves do not follow a repetitive pattern and there are no distinct P waves.

Atrial fibrillation has significant morbidity and associated health care costs. Many medical and nonmedical treatments exist to control atrial fibrillation. Catheter ablation of atrial fibrillation foci is a relatively new technology to manage atrial fibrillation in selected patients.

Minimally invasive techniques in all fields of surgery and medicine have blossomed with new technology and increased patient interest. Long-term results are lacking and new potential complications related to the new technologies are now being seen.

Catheter-based therapies for atrial fibrillation are part of the current guidelines recommended by major international cardiovascular societies.[1]

Since the initial report by Haïnsaguerre and colleagues[2] of pulmonary vein foci as initiators of atrial fibrillation in 1998, many developments have been made to ablate these foci to eradicate atrial fibrillation. Long-term follow-up and potential complications from many of these advanced techniques are unknown.

Circumferential pulmonary vein and left atrial ablation is reported to be effective for paroxysmal and chronic atrial fibrillation.[3-5] In the 2011 focused update on the management of patients with atrial fibrillation, the American College of Cardiology, American Heart Association, and the European Society of Cardiology continue to recommend ablation therapies to manage atrial fibrillation.[1]

The technique for catheter ablation varies by institution and among electrophysiologists. Commonly, the procedure is performed using transesophageal echocardiogram to rule out the presence of atrial thrombi and assess ventricular and valvular function. An electrode catheter in the coronary sinus is used to record left atrial electrical activity as well as for pacing. After transseptal puncture, patients are anticoagulated with the aim to maintain an activated clotting time of 300 seconds or greater. Different devices exist to create lesions around the pulmonary veins, usually 1 to 2 cm from their ostia, along the posterior left atrium or roof and along the mitral isthmus.[6] Catheter ablation for atrial fibrillation has a reported morbidity of approximately 6%.[7]

Major complications after catheter ablation for atrial fibrillation have been reported in the cardiology, radiology, and surgical literature.[8-12] Factors identified as risk factors for complications include multiple transseptal punctures, prolonged and complicated sheath, catheter manipulations, and aggressive attempts at transmural ablation (high energy and high contact forces). The most common complication reported are pulmonary vein stenosis (1.63%), pericardial

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Disclosures: Authors have nothing to disclose with regard to commercial support.
Received for publication Aug 26, 2012; accepted for publication Oct 15, 2012; available ahead of print Nov 12, 2012.
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J Thorac Cardiovasc Surg 2013;145:e9-13
0022-5223/$36.00

http://dx.doi.org/10.1016/j.jtcvs.2012.10.022